Health Consultation

Potential Health Impacts on the Georgetown Residential Neighborhood from Air Pollution Sources, Including the King County International Airport.
Seattle, King County, Washington

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Prepared by
The Washington State Department of Health
Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

FOREWORD

The Washington State Department of Health (DOH) has prepared this Health Consultation in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR is part of the U.S. Department of Health and Human Services and is the principal federal public health agency responsible for health issues related to hazardous waste. This Health Consultation was prepared in accordance with methodologies and guidelines developed by ATSDR.

The purpose of this Health Consultation is to identify and prevent harmful human health effects resulting from exposure to hazardous substances in the environment. The Health Consultation allows DOH to respond quickly to a request from concerned residents for health information on hazardous substances. It provides advice on specific public health issues. DOH evaluates sampling data collected from an industrial site, determines whether exposures have occurred or could occur, reports any potential harmful effects, and recommends actions to protect public health.

For additional information or questions regarding DOH, ATSDR or the contents of this Health Consultation, please call the Health Advisor who prepared this document:

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Background and Statement of Issues

On September 15, 1997, the Georgetown Crime Prevention and Community Council (GCPCC) petitioned the Agency for Toxic Substances and Disease Registry (ATSDR) to examine the potential impacts on human health that may result from exposures to jet fuel emissions in the Georgetown Neighborhood. Assistance with risk assessment preparations, based on community air sampling, was also requested. Due to their proximity to the to the King County International Airport (KCIA), locally referred to as Boeing Field, the GCPCC is particularly concerned about potential exposures to toxic substances attributable to emissions from KCIA. In 1998, ATSDR representatives in Atlanta, Georgia reviewed and analyzed data collected by The Washington State Department of Ecology (Ecology), Boeing, and GCPCC.[1] After review, ATSDR recommended that the Washington State Department of Health (DOH) develop a written health consultation to thoroughly evaluate the existing data, more completely define the issues which need to be addressed, identify additional community concerns, and determine necessary further actions. Using the available data, this health consultation evaluates potential human health effects resulting from the exposure to toxic air contaminants in the Georgetown residential neighborhood.

Site Background

Established in 1928, KCIA encompasses 594 acres within King County, Washington. Owned and managed by King County, the airport is a public use facility that serves a mix of Boeing aircraft, corporate, charter, air cargo, air taxi operations, and personal aviation activities. Trespassing at this site rarely occurs as the perimeter of KCIA is completely fenced and the facility is patrolled by security 24 hours per day.[2]

Located in a valley, approximately 17 feet above sea level, the airport is about 5 miles south of downtown Seattle and 5 miles north of the Seattle-Tacoma International Airport. KCIA is within the city limits of both Seattle and Tukwila. To the east of KCIA is the I-5 corridor and to the west is the Duwamish River. To the north is the Georgetown neighborhood, and to the south is the Allentown neighborhood. Both these neighborhoods are densely populated with mixed residential and urban/commercial zoning. The two runways at KCIA are in the north/south direction, making the Georgetown and Allentown communities the most heavily impacted by mobile source emissions, including overhead aircraft flying at lower elevations. Wind rose data at KCIA show that the predominant wind direction in this region is from the south and southeast, thus blowing towards the Georgetown neighborhood. A map of KCIA and the adjacent neighborhoods is shown in Appendix A.

Fifty-one percent of the property at KCIA is used for runways, ramps, and taxiways. The Boeing Company leases 21% of the total airport space (127 acres). The remainder of the facility is leased or rented to other corporations and individuals, and includes airport buildings, public roads, and parking areas. There are approximately 375,000 operations (takeoffs and landings) at the airport each year. Eighty-five percent of these operations are by helicopters and single and twin engine

planes. The remaining 15% of these operations are instrument operations (predominantly jets which are mostly commercial and cargo operators).[2, 3]

Community Concerns

On February 2, 1998, KCIA released a master plan that proposed shifting a runway 800 feet to the north.[3] Neighboring residents are concerned about the active runway being located closer to their homes. They are worried about the potential for adverse health effects that may be caused by jet engine exhaust.

Hospitalization rates, for all respiratory diseases in 1991 through 1995, for the Georgetown and South Park Communities (the 98108 zip code) have been significantly higher than in other King County areas for persons ages 0-64.[4] Hospitalization rates for asthma are also significantly higher than King County rates for persons ages 0-44.[4] While increased hospitalization rates may reflect higher rates of asthma and other illnesses in the 98108 zip code, they may also be associated with a lack of access to health care which could lead to higher numbers of hospital visits and increased hospitalization rates.[4] When compared to the overall Seattle averages, the Georgetown community, as well as other surrounding neighborhoods, has higher mortality rates and decreased life expectancies.[4] Residents are concerned about these elevated rates.

Volunteers at the Georgetown Powerplant Museum, located on the Northwest corner of KCIA, are also concerned about changes in the airport master plan.[5] As the Powerplant is a designated National Historic Landmark, there is concern that moving an airport runway closer to the Powerplant could have significant adverse impacts on the visitors and operations at the Powerplant Museum.[6]

Environmental Contamination

Using funding from their EPA Environmental Justice Grant, with assistance from multiple private consultants and public universities, GCPCC conducted sampling in and around the Georgetown neighborhood, KCIA, and Allentown. Although GCPCC, and their volunteers, were aided by professionals, the samples collected were not always collected and analyzed using the best available methodologies and techniques. For many samples, the exact collection methods are unknown and strict laboratory controls and standardized test protocols were not always employed. Therefore, the outcome of these sampling events may not represent what would be detected with monitoring and analysis conducted by certified professionals. Nevertheless, the data represent a good starting point to identify chemicals that might be of particular concern to GCPCC. Although the majority of the samples collected were air samples, GCPCC did have some drinking water and soil samples evaluated. All the data available from GCPCC will be presented in this section, but the health-based analysis will focus on the air data as inhalation exposure is the pathway of highest concern in this neighborhood.

Beginning in December 1996, GCPCC collected multiple air samples in the Georgetown neighborhood and one air sample in the Allentown neighborhood. Health concerns in the Allentown neighborhood are addressed in a larger study entitled: Addressing Community Health Concerns Around Sea-Tac Airport. Information regarding this ongoing study is available from the DOH Office of Epidemiology. Only contaminants detected in the Georgetown neighborhood will be discussed in this health consultation. The Oregon Graduate Institute of Science and Technology analyzed the collected samples. Although many hydrocarbons were detected, only three exceeded health-based screening guidelines. Health-based screening guidelines (also referred to as comparison values) are set by federal regulatory agencies such as ATSDR and EPA. These agencies use peer-reviewed scientific literature, in conjunction with conservative exposure assumptions, to derive health-based screening guidelines that will err on the side of health protection for the populations that are most at risk. In this health consultation, only contaminants detected at levels equal to or exceeding cancer and/or non-cancer screening values were further evaluated as contaminants of concern.

Contaminants of concern do not necessarily represent a public health hazard, but do warrant further investigation. Contaminants present at levels below health screening values are unlikely to have an adverse health impact. Contaminants of concern, the maximum detected levels, and their respective health-based screening values are shown in Table 1. These contaminants were detected at a residential street approximately 2-3 blocks from the north fence of KCIA.

Table 1. Maximum concentrations of contaminants detected in Georgetown in December 1996.

Contaminant	Maximum Concentration ($\mu g/m^3$)	Comparison Value ($\mu g/m^3$)	Source of Comparison Value
1,3-Butadiene	4.7	0.004	CREG
Benzene	44.6	0.1	CREG
1,3,5-Trimethylbenzene	9.8	6.2	PRG

CREG = ATSDR's Cancer Risk Evaluation Guide
PRG = EPA Region 9 Preliminary Remediation Goal
$\mu g/m^3$ = micrograms per cubic meter

In March and August of 1997, eight more air samples were collected by the GCPCC in the Georgetown neighborhood and between runways at KCIA. Samples were analyzed for hydrocarbons by the Washington State University Department of Civil and Environmental Engineering. The levels of air contaminants detected were much lower than the levels detected in samples analyzed in December of 1996. Benzene was detected at levels 10-fold lower than previously detected and 1,3,5-

trimethylbenzene levels were below the EPA Region 9 Preliminary Remediation Goal (PRG). These samples were not analyzed for 1,3-butadiene.

Air samples collected in March, 1997, were also analyzed by a private lab for SO_2 (sulfur dioxide), NO_x (nitrogen oxides), and aldehydes. The level of formaldehyde measured in the Georgetown neighborhood (4.8 parts per billion, ppb) exceeded the ATSDR health-based screening value of 3 ppb. Other compounds analyzed either were not detected by the analytical protocol used, or within safe levels.

Nine other air samples (tedlar bag, passive samplers, and charcoal tubes) were also collected by the GCPCC in the Georgetown neighborhood. Four samples were collected during August 1997, a fifth (passive sampler) was collected in October/November 1997, and the last sampler was used in November 1997 through January 1998. Charcoal tubes were used to collect samples in December 1998 and January 1999. A private lab analyzed these samples for volatile organic compounds (VOCs). All compounds detected were at levels below health-based screening values. A table of air sampling times, techniques, and results is shown in Appendix B. Unfortunately, information regarding the specifics of the sampling techniques and analytical procedures is limited.

A soil sample collected in August 1997 at a playfield in the Georgetown neighborhood was analyzed for VOCs and lead. VOCs were not detected. Lead levels were below Washington State health-based screening values. An August 1997 water sample collected from a faucet at a personal residence in Georgetown was also analyzed for VOCs. The only detected contaminant was chloroform and it was present at levels below health-based screening values.

In June 1998, soil samples from stockpiled and excavated soils were collected in the Northwest corner of the KCIA property, near the Georgetown Powerplant Museum. Soil collection was conducted by volunteers at the Georgetown Powerplant Museum. These soils were transported to this area by KCIA for aeration. Of the 69 samples collected, Total Petroleum Hydrocarbons (TPH) were detected in 16 samples at levels above the state recommended guidance for industrial soils as suggested by the Model Toxics Control Act (MTCA). There are many different TPH fractions, such as gasoline and diesel, and most have differing toxicities and levels of volatility. The particular TPH fractions detected in the soil samples have not been identified. KCIA has been unable to provide DOH with further information regarding these soils. Examination of these soils shows that they have been seeded. As of August 1999, grass covered the soils and they are more than 95% vegetated. Dust and inhalation exposures are therefore minimal. Additionally, as access on the KCIA property is controlled, contact with these soils by trespassers and the general public is unlikely; however, the Georgetown Powerplant museum is currently laying tracks for an outdoor miniature steam engine ride[5]. Due to this expansion, in the future more people, including children, are expected to spend time outside of the museum, but on the Powerplant property. For this reason, future aeration of soils should be monitored. If further information suggests that there may be the potential for exposure and subsequent health concerns, DOH will evaluate the data.

Potential Sources of Contamination

Jet engines are not the sole source of air contaminants detected in the Georgetown neighborhood. As with many urban and industrial areas, there is heavy automobile and truck traffic in Georgetown. The Georgetown community is home to many industrial sites including but not limited to: an aircraft painting facility, a chemical manufacturing company, a lamination business, and a specialty glass production company. There are also at least 16 companies subject to Toxics Release Inventory (TRI) reporting as required by the 1990 Pollution Prevention Act.[7]

To determine the impact KCIA has on the air quality in Georgetown, a combination of computer modeling (based on known releases) and air monitoring can be used. When using both these techniques it will be necessary to consider the meteorology, topography, and land uses in this area.

Discussion

Although it is very difficult to determine the sources of air contamination, using the limited data available, exposures were estimated and subsequently compared with health-based screening values and scientific literature for each of the contaminants of concern. 1,3-Butadiene, benzene, 1,3,5-trimethylbenzene, and formaldehyde were the only compounds detected at levels exceeding their respective comparison values. These contaminants were further evaluated to determine potential health risks and are discussed below. Please see Appendix D for a complete discussion of how the detected levels of contaminants compare to U.S. urban background air toxic levels.

1,3-Butadiene

1,3-Butadiene is a colorless gas with a mild gasoline-like odor. Some people can detect the odor of 1,3-butadiene at approximately 1 ppm (part per million).[8] Made from the processing of petroleum, 75% of the manufactured 1,3-butadiene is used to make synthetic rubber which is widely used for tires on cars and trucks. Environmental emissions of 1,3-butadiene come from motor vehicles and the burning, manufacture, transport, use, and disposal of fossil fuels.[8] 1,3-Butadiene is also emitted in tobacco smoke.[8] 1,3-Butadiene breaks down quickly in air (about 2 hours in sunlight) and evaporates easily. It is not expected to be found in soil and water.

A report on hazardous air pollution emissions in the Seattle-Tacoma urban area, published in 1995, found that mobile sources contributed to 88% of the 1,3-butadiene in the area.[9] Suburban/urban background levels of 1,3-butadiene often range from 0-6.5 ppb.[10] In 1977-1978, monitoring of 498 sites in the U.S. found the average to be 1.5 ppb but most other studies place the average air level at 0.3 ppb.[8,11] A recent study in Australia found that during peak hours on the freeway, 1,3-butadiene levels inside a new car can average 5.5 ppb. In an old car the average 1,3-butadiene level is 11.5 ppb.[12] In a smokey bar, 1,3-butadiene has been detected at 1.2 to 2 ppb.[13] The maximum level detected in the Georgetown neighborhood was 2.1 ppb (4.7 $\mu g/m^3$).

1,3-Butadiene and Cancer Risk

Cancer Risks

Cancer risk estimates are assumed to never reach zero, regardless of how minimal the exposure to a carcinogen may be. Terms used to describe this risk are defined below as the number of excess cancers that may be expected in a lifetime.

Term		# of Excess Cancers
Moderate	is approximately equal to	1 in 1,000
Low	is approximately equal to	1 in 10,000
Very low	is approximately equal to	1 in 100,000
Slight	is approximately equal to	1 in 1,000,000

Based on animal studies, 1,3-butadiene may be reasonably anticipated to be a carcinogen in humans.[8] EPA classifies 1,3-butadiene as a probable human carcinogen base on sufficient animal data and inadequate human data.[14] In animals, 1,3-butadiene has been shown to cause cancer in multiple organ systems, including the blood, lymph, and lung. Human epidemiological studies have looked at workers who were exposed to 1,3-butadiene in the rubber industry. These exposures were in the presence of many other compounds, which may also cause cancer, making a direct link between 1,3-butadiene and human cancer difficult to infer.[8]

In an animal study, tumors were seen throughout the body of rats exposed to 625 ppm 1,3-butadiene.[8] Mice were much more sensitive to 1,3-butadiene as tumors were noted after exposure to 6.25 ppm. When considering the toxicity and metabolism of 1,3-butadiene, humans are believed to be more closely related to the rat than the mouse.[8] ***In the Georgetown neighborhood, assuming that residents are consistently exposed to 2.1 ppb (4.7 $\mu g/m^3$) 1,3-butadiene, the risk of increased excess cancers is moderate (0.0013).*** A lifetime inhalation exposure at this level is estimated to result in 1.3 additional cancers per 1,000 persons exposed. This is a conservative assumption as 2.1 ppb was the highest level detected in the Georgetown community. Exposure assumptions and risk calculations are shown in Appendix C.

1,3-Butadiene and Non-cancer Risk

1,3-Butadiene acts as a mild narcotic in humans, causing lethargy and drowsiness at levels above 1000 ppm. It can also act as an eye, nose, and throat irritant when high level exposures occur. High exposure levels may also cause blurred vision, nausea, central nervous system damage, and unconsciousness.[8] Cardiovascular effects and effects on red blood cells have been seen in workers after exposure to 1,3-butadiene in conjunction with exposure to other chemicals.[15] ***At the 1,3-butadiene levels detected, 2.1 ppb, non-cancer health effects are not expected to occur.*** The non-cancer effects of 1,3-butadiene have only been documented at exposure levels at least 1000-fold greater than the level measured in the Georgetown neighborhood.

Benzene

Benzene is a colorless liquid with a sweet odor. Since benzene is highly volatile, inhalation is the dominant pathway for human exposure. Formed from both natural and man-made processes, benzene has been detected in air samples from rural and urban environments, and in indoor air. Gasoline vapors, auto exhaust, and chemical production are the main sources of benzene in the environment.[16] Benzene is widely used to make rubber, lubricants, dyes, detergents, and drugs. Benzene is also used in the production of plastics, resins, nylons, and synthetic fibers.[16] A natural component of cigarette smoke, crude oil, and gasoline, benzene is particularly important for unleaded gasoline due to its anti-knock characteristics. Unleaded gasoline contains approximately 1-2% benzene.[17]

Outdoor air contains low levels of benzene from tobacco smoke, automobile service stations, motor vehicle exhaust, and industrial emissions. Ambient air surrounding gas stations, refineries, and hazardous waste sites has higher benzene levels.[16] Benzene in indoor air comes from cigarette smoke, glues, paints, waxes, and detergents. In the Seattle-Tacoma area, excluding major stationary point sources, mobile sources were estimated to contribute to approximately 83% of the benzene released from stationary and mobile sources combined.[9] Riding in an automobile for 1 hour/day may lead to a benzene exposure of 40 μg/day.[18] Interestingly, EPA Total Exposure Assessment Monitoring studies have shown that for an individual, the most important sources of pollution are small and close to the person, and that exposures are not correlated with emissions.[16] Although over 80% of benzene in outdoor air comes from automobile exhaust, about half of the total national exposure to benzene comes from cigarette smoke.[19] A smoker who consumes 2 packs of cigarettes per day will have an additional daily benzene intake of 1,200 μg.[20]

Ambient air data, collected from thirty-nine U.S. urban areas in 1984-1986, found median benzene levels to be 40.25 $\mu g/m^3$ with a range of 15 $\mu g/m^3$ to 118 $\mu g/m^3$.[21] In the Georgetown neighborhood benzene was detected at 44.6 $\mu g/m^3$ (14 ppb), a value that is within the U.S. urban air range.

Benzene and Cancer Risk

The primary health effect of concern for benzene is leukemia, specifically acute myelocytic leukemia (AML).[16] This is the only health outcome that is consistently associated with occupational exposure to benzene. AML is not seen at exposure levels below 20 ppm-years. This would be equivalent to an average exposure of 2 ppm (parts per million) for a 10 year period. Many studies documenting human exposures have shown a link between benzene exposure and leukemia.[16] In most of these studies, the estimated exposure level was often above 100 ppm or within a broadly defined range (i.e., 10-100 ppm).[16] Lower level exposures (0.3-30 ppm) have also been associated with leukemia, but these workers were exposed to benzene as well as multiple other compounds present in a chemical manufacturing facility.[22] Another study of oil refinery workers, did not see a link between benzene and leukemia at lower concentrations (0.53 ppm).[23] Animal studies clearly support the

human

evidence and show a link between benzene exposure and leukemia. However, most of these studies exposed animals to benzene at levels above 100 ppm.[16]

Although the exposure level in the Georgetown community is lower than the exposures documented in previous occupational studies, benzene is still a contaminant of concern. Even though the level is within range of urban background levels within the U.S., high background levels do not diminish the potential for adverse health effects. ***In Georgetown, the risk of excess increased cancers is low (0.00035).*** A lifetime inhalation exposure to benzene, at the current detected level (44.6 $\mu g/m^3$, 14 ppb), is estimated to result in 3.5 additional cancers per 10,000 persons exposed. Exposure assumptions and risk calculations are shown in Appendix C.

Benzene and Non-cancer Risk

Brief exposure to very high levels of benzene in air (above 10,000 ppm) can result in death. Lower levels (700-3,000 ppm) can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness.[16] The effects from lower level exposures are reversed when the exposure to benzene is removed. Benzene can also suppress the immune system and disrupt normal blood production, leading to anemia and excessive bleeding.[16] In humans, changes in blood components have been documented at exposure levels as low as 3.2-12.8 ppm benzene for a 1-3 year duration.[16] Excessive benzene exposure may also be harmful to the reproductive organs. The levels of benzene that have been reported to cause its non-cancer effects are at least 200-fold greater than the levels detected in the Georgetown neighborhood (14 ppb). ***Non-cancerous adverse health effects are not expected to result from the levels of benzene that were detected.***

1,3,5-Trimethylbenzene

1,3,5-Trimethylbenzene is a component of several solvent mixtures that are used extensively for industrial purposes. It is often present in solvents used in paints and varnishes. Trimethylbenzenes occur naturally in coal tar and in many petroleums.[24] Trimethylbenzenes exist in the atmosphere as a gas, yet there are no data on current ambient air concentrations. Trimethylbenzene production occurs during petroleum refining and is a major component of the C9 aromatic hydrocarbon fraction.[25] Nearly 200 refineries in the U. S. produce this C9 fraction. Approximately 99% of the C9 fraction is then used as a gasoline additive.[25] Trimethylbenzenes (of which there are three isomers) make up approximately 30% of the C9 fraction.

1,3,5-Trimethylbenzene and Cancer Risk

There are little data on the effects of individual trimethylbenzene isomers. In general trimethylbenzene has not been associated with any carcinogenic outcomes. Studies of the

carcinogenic potential of trimethylbenzene have not been reported.[24] ***Although trimethylbenzene is unlikely to cause cancer, until it is proven to have only non-carcinogenic effects, the cancer risk to residents of Georgetown who are exposed to trimethylbenzene should be considered indeterminate.***

1,3,5-Trimethylbenzene and Non-cancer Risk

Although there is very little data on 1,3,5-trimethylbenzene, there is a some information on the non-cancer effects of 1,2,4-trimethylbenzene. All the current literature shows both isomers to have similar adverse effects.

At high doses (above 5000 ppm) 1,3,5-trimethylbenzene has been shown to be an eye, skin, and respiratory tract irritant.[25] Neurotoxic effects, respiratory diseases, blood disorders, and digestive disorders have also been associated with workers exposed to solvents containing trimethylbenzenes.[25] Workers exposed to a solvent (10-60 ppm) that contained 80% trimethylbenzenes showed symptoms of nervousness, anxiety, and asthmatic bronchitis. Animal studies have shown decreased weight gain and adverse blood changes at 1000 ppm. In animals, a no adverse effect level (NOAEL) for neurotoxic effects, including neurodegenerative changes, was shown to be 25 ppm.[25] Based on the general effects of trimethylbenzene, the Occupational Safety and Health Administration (OSHA) has set a occupational exposure level at 25 ppm for an 8 hr period. ***The level of trimethylbenzene in Georgetown, 9.8 $\mu g/m^3$ (2 ppb) is not expected to cause adverse non-cancer health effects.*** The level in the community, as measured, is 12,000-fold lower than the NOAEL in animal studies, and well below levels at which any adverse human health effects have been reported.

Formaldehyde

At room temperature, formaldehyde is a colorless and flammable gas that has a pungent and distinct odor. Formaldehyde is produced in small amounts everyday in all animals, including man, as part of normal metabolic processes.[26] This level of formaldehyde production causes us no harm. In the environment, combustion processes account for most of the formaldehyde in the air.[26] These combustion processes include: power plants, incinerators, refineries, wood stoves, cigarettes, tobacco products, and open fireplaces. Commonly found in many industries, formaldehyde is used in the production of fertilizers, paper, and plywood, and is also used in hospitals and laboratories to preserve tissues. Formaldehyde is also present in embalming fluid and is sometimes used as a food preservative. Formaldehyde can also be found in household products such as antiseptics, glues, cleaners, cosmetics, particle board and plywood cabinets, walls, and furniture.

The average outdoor air concentration of formaldehyde ranges from 2-6 ppb, with a mean of approximately 2.5 ppb. In industrial areas, levels can be 10-20 ppb.[26] A report on hazardous air pollution emissions in the Seattle-Tacoma urban area, published in 1995, found that mobile sources contributed to 74% of the formaldehyde in the area.[9] Typical daily outdoor exposures to

formaldehyde are 0.2-5 mg/day. Smoking 20 cigarettes increases this exposure by 1 mg/day.[26] Persons with the highest formaldehyde exposures live in mobile homes where formaldehyde is in the insulation and the structural plywood. In mobile homes, formaldehyde levels are in the range of 20-800 ppb. These levels are higher in new mobile homes.[26]

Formaldehyde and Cancer Risk

EPA classifies formaldehyde as a probable human carcinogen.[27] This is based upon limited human evidence and sufficient animal evidence. Multiple studies have shown a link between respiratory cancers and formaldehyde exposures in humans. These studies identified a cancer effect range as low as 0.1-10 ppm.[28] In animal studies, nasal cancers have also been seen after long-term inhalation of formaldehyde. A NOAEL for cancer in rats has been determined to be 2 ppm.[29] In Georgetown, formaldehyde was measured at 4.8 ppb (5.9 $\mu g/m^3$), a level above the U.S. average, but within the range of levels normally detected. ***In Georgetown, a very low increased cancer risk (0.000077) does appear to exist.*** A lifetime inhalation exposure to formaldehyde, at the current detected level, is estimated to result in 7.7 additional cancers per 100,000 persons exposed. Exposure assumptions and risk calculations are shown in Appendix C.

Formaldehyde and Non-cancer Risk

Common symptoms of exposure to formaldehyde include irritation of the eyes (including tearing), nose, and throat.[26] Formaldehyde also causes a burning sensation to the nose, eyes, and lungs. Formaldehyde is a direct irritant to the tissues it comes in contact with; since it is rapidly degraded, few effects are seen on tissues other than those directly contacted. Although people have differing sensitivities, most of these symptoms occur at exposures of 0.4-3 ppm. Asthmatics may be more sensitive to the effects of formaldehyde. Some studies have shown decreases in the lung function and increased bronchial reactivity of exposed asthmatics, but these studies are not consistent.[26]

Based upon studies showing injury to the nasal passages in humans, a low adverse effect level (LOAEL) for formaldehyde has been set at 0.98 ppm. ATSDR has set a minimal risk level, for chronic formaldehyde inhalation, at 3 ppb (3.7 $\mu g/m^3$). ***The level of formaldehyde, as measured in the Georgetown community (4.8 ppb), is not expected to cause adverse non-cancer health effects.***

Combined Cancer Risks for 1,3-Butadiene, Benzene, and Formaldehyde

When the cancer risks for the individual contaminants are added together, the cumulative cancer risk over a lifetime exposure would be considered moderate, with 1.7 excess cancers per 1,000 persons exposed. This value assumes that residents are chronically exposed to each contaminant at their maximum respective detected levels. This cancer risk is unacceptably large, however this estimated cancer risk is based on incomplete data. The available data does not allow us to determine

how often the air contaminants are at this maximum detected level. The values used in this health consultation may be representative of short-term or infrequent high exposures rather than long-term (chronic) average exposure levels. The adverse health outcome data for most contaminants are based upon a continuous long-term exposure period. Similar adverse effects may not be seen if the exposures are short and intermittent. Since the air samples were collected over short periods of time (usually a one hour period), the long-term average air levels of these contaminants are still unknown.

Chemical Exposure and Children

Children can be uniquely vulnerable to the hazardous effects of many environmental toxicants. When compared to adults, pound for pound of body weight, children drink more water, eat more food, and breathe more air. Children have a tendency to play closer to the ground and often put their fingers in their mouths. These factors lead to an increased exposure to toxicants in dust and soil. Additionally, before birth, the fetus is highly sensitive to many chemicals that may cause organ malformations and even premature death. For these reasons, it is very important to consider the specific impacts that contaminants may have on children, as well as other sensitive populations.

1,3-butadiene

In animal studies, 1,3-butadiene has been shown to cause miscarriages and birth defects.[8] The level at which no adverse developmental effects were seen in rats was 200 ppm. In mice, this no effect level is 20 ppm. Developmental effects include fetotoxicity and skeletal abnormalities. Reproductive effects, specifically ovarian atrophy, have been seen in mice after a chronic exposure (65 weeks) to 6.25 ppm 1,3-butadiene. In rats, the no effect level for developmental effects (after a ten day exposure during gestation) was 200 ppm. The no adverse effect level detected in mice is approximately 3,000-fold higher than the exposure level in Georgetown. The International Agency for Research on Cancer (IARC) has determined that the rat is an appropriate conservative model for the effects of 1,3-butadiene exposure.[8] The no adverse developmental effect level in rats is over 95,000-fold higher than the levels of 1,3-butadiene detected in Georgetown. ***It is therefore unlikely that 1,3-butadiene, at the levels detected, will have any adverse developmental or reproductive effects on persons living in the Georgetown community.***

Benzene

The effects of benzene on the developing fetus in pregnant women are not known, but a link between benzene exposure (greater than 1 ppm) and decreased female fertility has been suggested.[16] Animal studies show harmful effects including low birth weight, delayed bone formation, and bone marrow damage. These effects were seen at levels above 10 ppm (32 mg/m^3) when animals were exposed to benzene during the gestational period. The maximum level of benzene detected in the Georgetown neighborhood is 715-fold lower than this level.

Children may be at increased risk for the hematological (blood-based) effects of benzene. Blood cells that are still increasing in number and dividing are at greater risk than mature cells; as children are rapidly growing, they are at increased risk.[16] This increased risk may lead to aplastic anemia and possibly leukemia. As most documented benzene exposures occur in the workplace, there are no human studies specifically documenting the effects of benzene on children. Currently, the benzene levels in the Georgetown community exceed the cancer risk evaluation guideline (CREG) provided by ATSDR. ***Children in the Georgetown community might be at increased risk for both cancerous and non-cancerous adverse health effects caused by the current benzene levels.***

1, 3, 5-Trimethylbenzene

There is no information on the developmental or reproductive effects of trimethylbenzenes in humans.[25] Animal studies have looked at the developmental/reproductive effects of C9 hydrocarbons, of which trimethylbenzene is a large fraction. In animal studies, after exposure to 500 ppm of C9 hydrocarbons a decrease in maternal weight was seen. Another study on C9 hydrocarbons showed adverse developmental effects, including malformations and reduced viability, at 100 ppm.[25] These levels are far above the levels of trimethylbenzene measured in the Georgetown community. ***It is therefore unlikely that trimethylbenzene, at the exposure levels detected, will have any adverse developmental or reproductive effects on persons living in the Georgetown community.***

Formaldehyde

Adverse reproductive effects, as measured by a decreased sperm count, have been seen in men exposed to formaldehyde.[26] Based on this outcome, the NOAEL for the reproductive effects of formaldehyde is 1.32 ppm. Developmental effects have not been seen in humans, but an animal study has shown a NOAEL of 9.9 ppm based upon decreases in fetal weight. ***As the level of exposure in Georgetown is below the observed NOAELs for reproductive and developmental effects, formaldehyde, at the levels detected, would not be expected to cause any of these adverse effects.***

As formaldehyde can act as a respiratory sensitizer, and certain studies have shown children to be particularly sensitive to developing asthma, children might be at increased risk. Asthma levels in children are rising and children are particularly sensitive to many compounds that may trigger asthma or asthma attacks. Information on formaldehyde exposure and children is lacking. Studies on human health have looked at exposure concentrations above those detected in the Georgetown neighborhood. Since there is not sufficient data on children and formaldehyde and asthma, the risk to children in Georgetown is considered to be indeterminate.

Conclusions

Due to the lack of information about the quality control, the limited number, and the short time periods used for collection of the air samples, it is very difficult to draw firm conclusions regarding the potential for adverse human health effects associated with air pollution exposure in Georgetown. It is not known whether the levels of contaminants detected in Georgetown represent peak, low, or average levels, nor is it possible to determine if individuals in the community are continuously exposed to these levels. It is also not known whether KCIA is the source of these contaminants, as many other air pollution sources can be found in and near the Georgetown neighborhood. ***The detected exposure levels can only serve as rough estimates (which may or may not be representative of long-term exposure levels) until better monitoring and modeling data are collected***. Conclusions in this health consultation are based solely on the data presented in this document as it contains the only exposure information currently available.

An indeterminate public health hazard exists for persons living in the Georgetown community. This is due to the limitations and inadequacies of the available air sampling data. If further sampling confirms that the levels detected by the GCPCC represent chronic exposure levels, then the potential for adverse health effects may exist. This is predominantly due to the cancerous effects of 1,3-butadiene, benzene, and formaldehyde. As discussed in this health consultation, when the cancer risks for the individual contaminants are added together, the cumulative cancer risk for a lifetime exposure would be considered moderate. This risk has been determined assuming that the Georgetown residents are chronically exposed to the highest levels of each contaminant that was detected.

An indeterminate health hazard, based on cancerous effects, exists for Georgetown residents exposed to the measured levels of 1,3,5-trimethylbenzene. As the cancerous effects of 1,3,5-trimethylbenzene are currently unknown, it is impossible to say that zero or low risk exists. For this reason, the risk must be considered indeterminate.

At the present levels, there is no apparent public health hazard, based on non-cancerous effects, for Georgetown residents exposed to the measured levels of 1,3-butadiene, benzene, 1,3,5-trimethylbenzene and formaldehyde. The current levels are safely below levels that are expected to cause adverse non-cancerous health effects. However, the data are incomplete.

For children in the Georgetown neighborhood, the public health hazard is indeterminate. This is due to the limitations and inadequacies of the available air sampling data***. There is the potential for adverse health outcomes based upon the cancerous and non-cancerous effects of benzene, but further air monitoring is necessary to determine actual exposure levels.***

An indeterminate health hazard also exists for children due to the non-cancerous effects of formaldehyde. Children, due to their size and their development are often a most sensitive population when exposed to environmental contaminants. This sensitivity creates the potential for children to be at increased risk for adverse health effects. As the specific hazards associated with exposure to formaldehyde cannot be determined, they are considered to be indeterminate.

The cancer risks to residents in the Georgetown community have been compared to the average U.S. urban background levels. These comparisons, and limitations of the available data, are discussed in Appendix D.

Recommendations

- Further air monitoring is necessary in the Georgetown neighborhood to confirm that the levels detected by the GCPCC are representative of the actual exposure levels. Appropriate control samples, sampling strategies, collection procedures, and analytical methods are necessary. EPA sampling methodologies should be used and analysis should include a full inventory of air contaminants. All potential sources need to be considered. Short-term sampling during aircraft testing and long-term sampling in the community should be conducted to ensure that the exposures can be more accurately estimated.

- Modeling is necessary to determine the source and movement of detected air contaminants. This should be conducted with the use of meteorological data and emissions inventories for the specific industries, including KCIA, in the Georgetown vicinity. The Puget Sound Clean Air Agency (PSCAA) should be able to provide emissions inventory data. Multiple source modeling using EPA-accepted models, such as ISCT-3, is recommended.

- To address the concerns of the GCPCC, specific and distinct attempts should be made to identify the impacts of KCIA, jet aircraft testing (including emissions from auxiliary power units), and aircraft landings, takeoffs, and run-ups on the community. Monitoring, sampling, and modeling that focus specifically on these impacts should be completely addressed.

    ⇨ **Actions:** An interagency task force has already been established to do a KCIA Air Quality Study. This task force includes representatives from EPA, Ecology, ATSDR, PSCAA, Boeing, the King County Department of Health, the Federal Aviation Administration, and community members. It is hoped that through this task force, a thorough study and characterization of air pollutants in the Georgetown vicinity will be conducted. This study should include computer modeling as well as sampling and monitoring in and around the KCIA. Air emissions inventories should also be collected. DOH will be playing a role on this task force.

    ⇨ **Actions:** DOH will review the output from all sampling, monitoring, and modeling to further evaluate the potential for adverse human health effects in the Georgetown community. Health-based evaluations, specifically addressing human health outcomes, will be conducted as this data becomes available.

- Future aeration of soils on the KCIA property should be monitored and/or regulated by the appropriate agency(s) to ensure that on-site emissions do not pose a health hazard.

- Because proximity to emission sources is directly related to adverse health outcomes, potential health impacts should be considered when deciding upon future land use practices, policies, and changes.

CERTIFICATION

This Health Consultation for the King County International Airport was prepared by the Washington State Department of Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Health Consultation was initiated.

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Technical Project Officer
Superfund Site Assessment Branch (SSAB)
Division of Health Assessment and Consultation (DHAC)

The Division of Health Assessment and Consultation (DHAC), ATSDR, has reviewed this Health Consultation and concurs with its findings.

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References

1. Zarus, Gregory. ATSDR Record of Activity LOG # 98-1042. Feb. 1998.

2. Impett, Clare, KCIA staff, personal conversation, June 1999.

3. KCIA, Master Plan Proposed Activities, Jan. 1997.

4. Seattle King County Department of Public Health, Hospitalization Rates for Respiratory Diseases by Age. June 1997.

5. Tellefson, Lilly, Georgetown Powerplant Museum, personal conversation, August 1999.

6. Sundberg, Charles, King County Office of Cultural Resources, letter to KCIA, October 31,1997.

7. Washington State Department of Ecology, Toxics Release Inventory, Publication # 98-402. June, 1998.

8. Agency for Toxic Substances and Disease Registry, Toxicological Profile for 1,3-Butadiene. Publication # TP-91/07, July 1992.

9. Mangino, J., Jones, JW, Mobile Source Hazardous Air Pollutant Emissions in the Seattle-Tacoma Urban Area. Govt Reports Announcements and Index, issue 10, NTIS/PB95-174660, 1995.

10. Hazardous Substances Data Base, 1,3-Butadiene National Library of Medicine Data Network, June 1999.

11. Brodzinsky, R., Singh, HB, Volatile Organic Chemicals in the Atmosphere: An Assessment of Available Data. Menlo Park, VA: Atmospheric Science Center, SRI International, 1992.

12. Duffy, BL, Nelson, PF, Exposure to Emissions of 1,3-Butadiene and Benzene in the Cabins of Moving Motor Vehicles and Buses in Sydney, Australia. Atmospheric Environments, 31: 3877-3885, 1997.

13. Brunnemann, KD, Kagan, MR, Cox, JE, et al. Analysis of 1,3-Butadiene and Other Selected Gas-Phase Components in Cigarette Mainstream and Sidestream Smoke by Gas Chromatography-Mass Selective Detection. Carcinogenesis 11:1863-1868, 1990.

14. Environmental Protection Agency, Integrated Risk Information System (IRIS) 1,3-Butadiene Feb. 1991.

15. McMichael, AJ, Spirtas, R, Gamble, JF, Tousey, PM, Mortality Among Rubber Workers: Relationships to Specific Jobs. J. Occup. Med. 18:178-185, 1976.

16. Agency for Toxic Substances and Disease Registry, Toxicological Profile for Benzene, Sept. 1997.

17. NESCAUM Evaluation of Health Effects from Exposure to Gasoline and Gasoline Vapors Final Report. Northeast Sates for Coordinated Air Use Management, Air Toxics Committee. 3-1-3-16, 51-5-33. 1989.

18. Wallace, LA, Major Sources of Benzene Exposure. Environ. Health Perspect., 82:165-169, 1989.

19. Wallace, LA, Human Exposure to Environmental Pollutants: A Decade of Experience. Clinical and Exp. Allergy, 25:4-9, 1995.

20. Fishbein, L. Exposure from Occupational Versus other Sources. Scand. J. Work Environ. Health, 18:5-16, 1992.

21. Environmental Protection Agency, Ambient Air Benzene Concentrations in 39 U.S. Cities, 1984-1986, EPA/600/d-87/160, 1987.

22. Ott, MG, Townsend, JC, Fishbeck, WA et al, Mortality Among Workers Occupationally Exposed To Benzene. Arch. Environ. Health, 33:3-10, 1978.

23. Tsai, SP, Wen, CP, Weiss, NS et al, Retrospective Mortality and Medical Surveillance Studies of Workers in Benzene Areas of Refineries. J. Occup. Med. 25:685-692, 1983.

24. Hazardous Substances Data Base, 1,2,4-Trimethylbenzene National Library of Medicine Data Network, 1995.

25. Environmental Protection Agency, Chemical Summary for 1,2,4-Trimethylbenzene. OPPT, 749-F-94-022a, 1994.

26. Agency for Toxic Substances and Disease Registry, Toxicological Profile for Formaldehyde, Sept. 1997.

27. Environmental Protection Agency, Integrated Risk Information System (IRIS) Formaldehyde, May 1991.

28. Stayner, LT, Elliott, L, Blade, L, Keenlyside, R, Halperin, W, A Retrospective Cohort Mortality Study of Workers Exposed to Formaldehyde in the Garment Industry. Am J. Ind. Med. 13:667-682, 1988.

29. Kerns, WD, Pavkov, KL, Donofrio, DJ, Gralla, EJ, Swenberg, JA, Carcinogenicity of Formaldehyde in Rats and Mice after Long-term Inhalation Exposure. Cancer Res. 43:4382-4392, 1983.

Appendices

A. Map of KCIA and Adjacent Neighborhoods

INSERT THE COLORED MAP HERE!!!!!!

B. Table of Air Sampling Events

Table of air sampling data collected by the GCPCC. Only contaminants of concern are shown. Comments represent notes taken by persons conducting the sampling and laboratories which performed sample analysis.

				Concentration in ug/m3				
date	method	time	location	benzene	1,3-butadiene	formaldehyde	1,3,5-trimethylbenzene	comments
12/18/96	TO14 canister #1	7:15-8:15	6420 Carleton*	44.6	4.7		9.8	aircraft departing
12/18/96	TO14 canister #2	7:22-8:22	13th Ave S. & Hardy*	30.4	4		6.1	aircraft departing
12/18/96	TO14 canister #3	7:22-8:22	Allentown	49.8	5.9		12	aircraft landing, diesel truck idling approx. 25m l to north for 10 min.
12/30/96	TO14 canister #5	20:25-21:45	6435 Flora*	5.6	.		1.6	planes departing, strong kerosene smell
3/4/97	TO14 canister #6	11:05-12:27	6428 carleton*	5.42			6.87	before 777 test
3/4/97	TO14 canister #7	12:45-14:26	power plant, KCIA*	10.53			19.63	during 777 test
3/4/97	TO14 canister #8	12:45-14:40	6428 carleton*	7.34			4.4	during 777 test
3/5/97	TO14 canister #9	4:35-6:26	KCIA runways*	10.2			1.96	
3/4/97	DNPH tubes	12:45-14:36	Georgetown*			6		during engine testing
7/24/97	TO14 canister #10	6:45-8:15	6435 Flora*	9.57			1.96	planes departing
7/25/97	TO14 canister #11	7:28-8:58	6435 Flora*	13.08			2.45	planes departing
7/25/97	TO14 canister #12	15:20-16:40	6435 Flora*	0.6			1.47	planes departing
7/25/97	TO14 canister #13	6:35-8:05	6435 Flora*	11.48			2.45	planes landing
8/7/97	tedlar air bag	17:31-17:36	6435 Flora*	nd			nd	detection level 0.09 ug/m3
8/14/97	tedlar air bag	16:35-16:40	6435 Flora*	nd			nd	detection level 0.09 ug/m3
8/14/97	passive air sampler	Aug 14-Sept. 15	6435 Flora*	nd			nd	detection limit 0.8 ppb
9/15/97	passive air sampler	Sept. 15-Oct.15	6435 Flora*	nd			nd	detection limit 0.65 ppb
10/15/97	passive air sampler	Oct 15-Nov 16	6435 Flora*	nd			nd	detection limit 2 ug/m3
11/16/97	passive air sampler	Nov.16-Jan 17	6435 Flora*	nd			nd	detection limit 2 ug/m3
12/23/98	charcoal tube	16:45-19:45	6435 Flora*	nd			nd	detection limit 2 ug/m3
12/23/98	charcoal tube	13:35-16:35	6435 Flora*	nd			nd	detection limit 2 ug/m3
1/15/99	charcoal tube	19:59-21:30	6435 Flora*	nd			nd	strong jet fuel odor, boeing runups occuring, detect 2 ug/m3

* these location are in the Georgetown Neighborhood
nd = not detected

C. Exposure Assumptions

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The only exposure pathway of concern in this health consultation is through the inhalation route. Using EPA and ATSDR risk assessment guidelines, no dose calculations are necessary for inhalation exposures. This is because the Minimal Risk Levels and Environmental Risk Evaluation Guidelines are listed as concentrations. The inhalation unit risk is listed as a risk for excess cancer per unit concentration. This value is derived by EPA using data from human exposures and animal studies. Therefore, for inhalation:

Risk = Air Concentration x Unit Risk

The unit risk values used in this health consultation are shown in the table below.

Contaminant	**Unit Risk** $(\mu g/m^3)^{-1}$
1,3-Butadiene	0.00028
Benzene	0.0000078
1,3,5-Trimethylbenzene	ND
Formaldehyde	0.000013

ND = not determined

D. Cancer Risk Evaluation: Limitations and Comparisons

To add perspective to the health-based exposure analysis, where sufficient literature was available, cancer risks for carcinogens detected in the Georgetown community were compared to average U.S. urban background levels. Limitations of the data set and ways to better collect information for a health-based exposure analysis are also discussed.

1,3-Butadiene

In the Georgetown neighborhood, assuming that residents are consistently exposed to 2.1 ppb (4.7 ug/m^3) 1,3-butadiene, the risk of increased excess cancers is moderate (0.0013). A lifetime inhalation exposure at this level is estimated to result in 1.3 additional cancers per 1,000 persons exposed. This is a conservative assumption as 2.1 ppb was the highest level detected in the Georgetown community. At the average U.S. background levels (0.3 ppb 1,3-butadiene) the cancer risk is 1.8 additional cancers per 10,000 persons exposed. Exposure assumptions and risk calculations are shown in Appendix C.

Benzene

In Georgetown, the risk of excess increased cancers is low (0.00035). A lifetime inhalation exposure to benzene, at the current detected level (44.6 $\mu g/m^3$), is estimated to result in 3.5 additional cancers per 10,000 persons exposed. At the average U.S. background levels (40.25 $\mu g/m^3$ benzene) the cancer risk is 3.1 additional cancers per 10,000 persons exposed.[16] Exposure assumptions and risk calculations are shown in Appendix C.

Combined Cancer Risks

When the cancer risks for the individual contaminants are added together, the cumulative cancer risk, over a lifetime exposure, would be considered moderate, with 1.7 excess cancers per 1,000 persons exposed. This cancer risk is unacceptably large, but from the sampling data available it cannot be determined how well the collected data represent the actual long-term exposure levels.

For perspective on the risk analysis numbers, if the background risks observed in the average U.S. urban area for the three contaminants of concern are added together, the cumulative cancer risk would be 5.7 excess cancers per 10,000 persons exposed. As measured, the risk in Georgetown is three-fold greater than the average U.S. background risk. This difference may be real, or it may be due to differences in sampling techniques. Most of the studies that were used to determine the U.S. background air contaminant levels employed continuous monitoring protocols and sampled for periods that extended from months to years. These studies also utilized highly specialized sampling equipment and very stringent protocols. For each contaminant of concern in Georgetown, the level

detected was within the range of regularly detected U.S. urban background levels. The difference between the Georgetown community and the U.S. urban average is less than one order of magnitude, and probably not considered to be significant given the uncertainties involved in risk assessment and in the sampling and data collection procedures that were used.

As discussed above, in Georgetown, the detected levels for each contaminant are within the range of U.S. average urban background levels. Although this means that millions of U.S. citizens are probably exposed to similar cancer risks, this does not diminish public health concerns. The high background levels of these contaminants does make it very difficult to determine the specific source(s) of contamination. It is possible that what was measured in Georgetown can be attributed simply to urban background levels caused by light industry and mobile source emissions. With the current data, it is impossible to determine what portion of the air pollution is due to KCIA or any other source.

Previous analysis, by ATSDR, correlated the measured benzene and 1,3-butadiene levels with Boeing 777 jet engine testing. Assuming that the contaminants were emitted from the jet engines, using data on the frequency and duration of jet engine testing, the average daily emission concentrations were calculated.[1] With these assumptions, the cumulative excess cancer risk for benzene and 1,3-butadiene, over a lifetime exposure, would be 7.4 excess cancers per 100,000 persons exposed.[1] This is considered to be a very low excess cancer risk. This scenario, and associated exposure assumptions, needs to be validated by further monitoring and computer modeling. Whether or not the levels detected represent peak concentrations, or typical long-term exposure levels, needs to be determined.

Although monitoring will confirm the presence of the contaminants detected, it will not be sufficient to determine the predominant sources of the individual air pollutants. It will also be necessary to collect emissions inventories for industries in the area. Once this data is collected, computer modeling should be used to determine the relative contribution of each identified source. Through the use of source specific data, computer models will allow predictions as to what portion of contaminants present are coming from KCIA, mobile source emissions, or other industries located in Georgetown. Based on what is currently known about the area, EPA-approved computer models such as Industrial Source Complex Terrain 3 (ISCT-3), or AERMOD should be useful for this purpose.

Glossary

Acute	Occurring over a short period of time. An acute exposure is one which lasts for less than 2 weeks.
Agency for Toxic Substances and Disease Registry (ATSDR)	The principal federal pubic health agency involved with hazardous waste issues, responsible for preventing or reducing the harmful effects of exposure to hazardous substances on human health and quality of life. ATSDR is part of the U.S. Department of Health and Human Services.
Carcinogen	Any substance that can cause or contribute to the production of cancer.
Chronic	A long period of time. A chronic exposure is one which lasts for a year or longer.
Comparison value	A concentration of a chemical in soil, air or water that, if exceeded, requires further evaluation as a contaminant of potential health concern. The terms comparison value and screening level are often used synonymously.
Contaminant	Any chemical that exists in the environment or living organisms that is not normally found there.
Cancer Risk Evaluation Guide (CREG)	The concentration of a chemical in air, soil or water that is expected to cause no more than one excess cancer in a million persons exposed over a lifetime. The CREG is a *comparison value* used to select contaminants of potential health concern and is based on the *cancer slope factor* (CSF).
Dose	A dose is the amount of a substance that gets into the body through ingestion, skin absorption or inhalation. It is calculated per kilogram of body weight per day.
U.S. Environmental Protection Agency (EPA)	Established in 1970 to bring together parts of various government agencies involved with the control of pollution.

Exposure	Contact with a chemical by swallowing, by breathing, or by direct contact (such as through the skin or eyes). Exposure may be short term (acute) or long term (chronic).
Hazardous substance	Any material that poses a threat to public health and/or the environment. Typical hazardous substances are materials that are toxic, corrosive, ignitable, explosive, or chemically reactive.
Lowest Observed Adverse Effect Level (LOAEL)	LOAEL's have been classified into "less serious" or "serious" effects. In dose-response experiments, the lowest exposure level at which there are statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control.
Maximum Contaminant Level (MCL)	A drinking water regulation established by the federal Safe Drinking Water Act. It is the maximum permissible concentration of a contaminant in water that is delivered to the free flowing outlet of the ultimate user of a public water system. MCLs are enforceable standards.
Model Toxics Control Act (MTCA)	The hazardous waste cleanup law for Washington State.
No apparent public health hazard	Sites where human exposure to contaminated media is occurring or has occurred in the past, but the exposure is below a level of health hazard.
No Observed Adverse Effect Level (NOAEL)	The dose of a chemical at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be observed at this dose but were judged not to be "adverse."
Organic	Compounds composed of carbon, including materials such as solvents, oils, and pesticides which are not easily dissolved in water.
Parts per billion (ppb)/Parts per million (ppm)	Units commonly used to express low concentrations of contaminants. For example, 1 ounce of trichloroethylene (TCE) in 1 million ounces of water is 1 ppm. 1 ounce of TCE in 1 billion ounces of water is 1 ppb. If one drop of TCE is mixed in a competition size swimming pool, the water will contain about 1 ppb of TCE.
Indeterminate public health hazard	Sites for which no conclusions about public health hazard can be made because data are lacking.

Risk The probability that something will cause injury, linked with the potential severity of that injury. Risk is usually indicated by how many extra cancers may appear in a group of people who are exposed to a particular substance at a given concentration, in a particular pathway, and for a specified period of time. For example, a 1%, or 1 in 100 risk indicates that for 100 people who may be exposed, 1 person may experience cancer as a result of the exposure.